On January 5th, 1881, the patient, a strong, healthy German woman, æt. 47, while lying upon her left side, in bed, was approached suddenly by her drunken husband and stabbed in the neck with a long narrow-bladed pair of shears; turning and endeavouring to rise, she received a second blow upon the ramus of the lower jaw, causing a long lacerated wound. Attempting to scream aloud for help, she found herself voiceless. On April 5th I was consulted on account of the aphonia, which had persisted since the injury.

Examination.—A long, irregular cicatrix on the border of the lower jaw, right side, a short distance from its angle; a second small one, evidently the result of a punctured wound, at the inner border of the sterno-cleido-mastoid muscle, at the level of the lower border of the cricoid cartilage on the same side. Patient completely aphonic, speaking only in a whisper, and suffering from slight dyspnœa, especially on exertion. The laryngoscope showed absolute paralysis of all of the muscles of the right vocal cord, it being fixed in the cadaveric position, *e. g.*, midway between the extremes of adduction and abduction, and motionless on attempted phonation or inspiration. The left vocal cord moves freely, and compensates for the defective action of its fellow by passing the median line on adduction, its arytenoid cartilage passing in front of that of the paralyzed cord, and thus fairly approximating the edges of the cords. Laryngeal mucous membrane normal.

Remarks.—The condition is of course an incurable one; the only possible error in diagnosis lies in default of a laryngoscopic inspection, in the supposition that the case was one of functional paralysis due to sudden emotion and terror. This theory is at once disproven by the direct examination of the condition of the vocal cords. That a clumsy instrument, roughly used, should have escaped wounding the vital vessels which it must have closely passed in its inward course, and severed alone a nerve the size of the recurrent lying in a protected situation, is hardly comprehensible. The fact, however, stands.

ARTICLE XVIII.

ON SOME OF THE CONDITIONS AFFECTING THE ORIGIN AND COURSE OF PULMONARY PHTHISIS.

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THE conditions affecting the origin and course of pulmonary phthisis are so numerous that it would be impossible, in the limits of a journal article, to properly consider even the majority of them. In the following pages I desire to call attention to three of them, to which my individual experience has directed my attention, viz.:—

I. Inflammation of the respiratory organs as they affect the origin and course of pulmonary phthisis.
II. Syphilis in its relations to the origin and course of pulmonary phthisis.
III. Contagion and inoculation.

These subjects are especially selected on account of their great importance and actual interest.

I. *Inflammation of the respiratory organs as they affect the origin and course of pulmonary phthisis.*

In this connection I would first direct attention to the influence of (*a*) laryngitis and bronchitis. I know full well, and notwithstanding the ancient views of Portal and Borsieri, who contended that pulmonary phthisis was frequently due to an ordinary cold, that this view has since that time been warmly debated. Louis, in his researches upon pulmonary phthisis, where the numerical method is so exhaustively adopted, endeavours to show by the thorough analysis of eighty cases that bronchitis has no direct effect in producing pulmonary phthisis. Since Louis' day students and practitioners have become familiar with Niemeyer's conclusions, who recognizes in bronchitis a direct and very efficient condition of the origin and progress of pulmonary phthisis. Amongst them he states that it extends its march to the pulmonary parenchyma, and that an acute broncho-pneumonia thus produced is too apt to lead to cheesy nodules which rapidly soften and form cavities occasioning after this manner an advanced stage of pneumonic phthisis. These conclusions are strongly combated by Flint in his late remarkable treatise on "Pulmonary Phthisis." If I refer to my own notes, I find that I am forcibly obliged to adopt the view that bronchitis, but more particularly laryngo-bronchitis, is an efficient and direct condition of the origin and course of pulmonary phthisis. In my experience, and upon close questioning, I find many patients who date the origin of their pulmonary affection to taking "cold." This cold has frequently been accompanied with some hoarseness and cough, which instead of disappearing rapidly and completely, remained and became aggravated and more distressing as time elapsed. In a fair proportion of cases I have been unable to trace the persistence of the hoarseness and cough, either to hereditary influences, or bad hygienic conditions. True it is, however, that an occasional and efficient condition of recurrence of an attack of laryngitis or bronchitis in its primary intensity was taking a fresh "cold," and this succession of acute attacks was obviously most detrimental to pulmonary integrity. Whenever the inflammatory change has seated itself within the larynx, it has almost invariably augmented the frequency and intensity of cough, and in a small degree increased the quantity of sputa. I have likewise remarked that such cases are more depressing to the general condition than those in which the inflammation is limited to the bronchial mucous membrane. Besides the increased cough, dysphagia, which accompanied the laryngitis at times, was seemingly the reason why little nutriment was taken, and thus helped along rapid diminution of flesh and strength. No doubt in numerous instances the laryngitis followed upon the bronchitis, or was at most an unimportant dependent of it, but there were numerous examples in which it appeared to me that but for the laryngitis the bronchitis would have had a much better chance of cure. Following directly upon the laryngo-bronchitis, I have clinically observed the advent of dulness at the apices with harsh breathing and more numerous râles at this region. These physical signs when combined with grave general symptoms left me but one probable belief, viz., that the lung had become consolidated in its upper portion and that this consolidation was most probably occasioned in the manner referred to.

(*b*) The inflammatory condition of the pulmonary parenchyma, such as we encounter in lobar pneumonia, has very rarely appeared to me to affect the origin and course of pulmonary phthisis. This is particularly true in almost all instances where the pneumonia attacked an individual previously healthy. Usually when he had recovered from this acute disease, cough and expectoration entirely disappeared; previous strength and flesh were regained, and shortly the patient was seemingly as well as before. At times, and when a whole lobe or lung became consolidated by reason of acute inflammation in a patient presumably phthisical already, it has obviously been an efficient condition of the more rapid advance of the phthisical disease of the lungs. True it is, however, that broncho-pneumonia, whilst it is more readily and frequently produced by the presence of miliary tubercle than the lobar form, is also more likely to lead to its manifestation and hasten its march. Nothing indeed is more insidious at times than the development of small nodules of consolidation of this kind in the lungs. If they are situated centrally, a slight rise of temperature may be almost the only symptom indicative of their existence. As they extend towards the surface of the lungs, or fresh nodules form more superficially, physical signs may clearly prove their existence. In very many examples of course both the local and general phenomena present, are sufficiently characteristic to enable us to affirm their development. Now, then, these nodules, instead of resolving and disappearing completely, will oftentimes become still more solid and cheesy, and around their outer limits too frequently do we recognize at the autopsy numerous miliary tubercles which evidently were under their dependence and proceeded directly from them by auto-inoculation carried on through the vascular supply of the affected region. It may be affirmed that it required a predisposing constitutional state to permit such effects to be produced. Too frequently, I am confident, no such hypothesis is warranted; and the local infiltration of broncho-pneumonia once established marches inevitably towards phthisical development in the pulmonary structure. In this opinion I hold to what the pathology of to-day certainly teaches, viz., that whenever catarrhal products become impacted in the smaller bronchi the pulmonary lobules are apt to become degenerated, general infiltration of pulmonary fibrous tissue takes place, and broncho-pneumonia, leading often to pulmonary phthisis, is developed. It is not essential, moreover, to believe with Watson that "dormant predisposition is awakened into actual disease and latent tubercles are accelerated" by intercurrent inflammation of pulmonary tissue.

(c) The influence of pleurisy upon the origin and course of pulmonary phthisis has been variously determined by different authors. While some see in the existence of a previous or present pleuritic inflammation a very direct and powerful condition of the development of pulmonary phthisis, as many other writers contend that the pleurisy is usually produced by the miliary tubercles already present, sometimes in its layers, sometimes immediately beneath them. Certain it is, as Louis has affirmed, and as almost all autopsies show, the most frequent pathological accompaniment of pulmonary phthisis is pleuritic adhesions. These adhesions may be partial or general. They may consist, indeed, only of a few bands, or fibres, connecting the two pleural surfaces together. Still they almost invariably are found in a greater or less extent of the pulmonary surface. The warmest advocate of their causative influence in producing pulmonary phthisis in the last few years has been Dr. J. R. Leaming, of New York, and he has made a new division of pulmonary phthisis based mainly on this conviction. To such a belief I can but oppose the fact that miliary tubercles are not infrequently seen within or upon the surface of the pleura, and, notwithstanding their presence, no pleuritic inflammation has been established. If we take into account, however, the frequency of pleuritic adhesions localized at the apices of the lungs, and the fact that these adhesions have obviously been the cause at times of pain and certain stethoscopic signs, when pulmonary respiration at their level was still vesicular and uncommonly pure—that following occasionally upon these signs, and after the lapse of several months, the evidences of adhesions remaining, cough becomes more frequent and harassing, flesh and strength are notably diminished, and roughened breathing with stationary small moist râles are discovered, all indicating the presence of pulmonary phthisis—how can we fail to recognize that pleuritis is a modifying agent of great importance as regards the origin of pulmonary phthisis? Whenever pulmonary phthisis is already developed, and pleuritic adhesions are added to it, there is little reason to doubt that they hasten very materially the onward and downward march of the former. When not yet produced, but imminently threatened, they effect this development, doubtless, in a very analogous way to what we recognize when other serous membranes become inflamed, or thickened, as, for example, when peritonitis is a primary condition of degenerative changes in the intestines, or pericarditis precedes a somewhat similar alteration in the connective tissue and muscular fibre of the cardiac walls. By their presence, chronic congestion and hyperplasia of the pulmonary tissue are obviously occasioned, being directly due to their contraction and to the pressure effects thus produced. In these changes further degeneration is liable to occur, and crude tubercle often originates.

II. *Syphilis in its relations to the origin and course of pulmonary phthisis.*

As is well known, the belief that pulmonary phthisis may be directly caused by syphilis is no new one. As far back as Morgagni such an opinion was sustained, and so late as the days of Laennec and Andral the notion was a prevalent one that phthisis was a not infrequent result of syphilitic infection. At this period it was held that the other pulmonary diseases which to-day we recognize as being distinctly syphilitic in their causation were intercurrent affections of quite different origin.

How does the question stand in our day? Is there or is there not a pulmonary phthisis which is caused directly by syphilis? Now, if by phthisis we mean tuberculosis, I answer in the negative; but if by phthisis we mean merely a wasting febrile disease, ulcerative in character, affecting particularly the lung-structures, why then I answer that there is such an affection. If the former view be held—namely, that all phthisis is tubercular—then the influence of syphilis in its etiology is but that of a predisposing condition. Syphilis, like most general diseases, impairs the nutrition of the economy, reduces the strength of the individual below the normal, and, finally, when the dyscrasia is firmly seated, becomes doubtless an important factor in the development of lung-disease, phthisical and even tubercular in nature. As regards the latter tenet, we know already and without question that, in the tertiary period of syphilis, interstitial pneumonia and gummy deposits are not uncommonly found in the lung-tissue. Depaul made the discovery in 1851. Since that time, Virchow, Weber, Lancereaux, Lebert, Moxon, Fournier, Randohr, Gamberini, and others have confirmed and extended our knowledge of these and other specific pathological conditions. Oftentimes, at the autopsies of syphilitic subjects, cavities have been found in different portions of the lungs, surrounded by walls of fibrous tissue, and presenting to the naked eye and to microscopic examination characters not dissimilar to those found in cadavers of persons who have succumbed to tubercular disease. And yet there are some distinguishing features, it is said, from a pathological standpoint: The nodules indicative of syphilitic changes are situated particularly in the lower lobes. They are less numerous and of larger size than the cheesy nodules of tubercular phthisis. Their colour and consistency are also signs by which they may be at times differentiated. The gummata are white or yellow, never transparent like miliary tubercle. They are of more uniform structure, and, if they break down, their destruction, on account of the nature of their capsule, is never so complete as that of a tubercular deposit. (Fournier, quoted by Bumstead, Venereal Diseases, 4th ed., p. 629.)

Whilst the above characters exist at times after death, and will then enable us to distinguish syphilitic pulmonary disease from that which is of tubercular nature, they are not always present. Prof. Gamberini (*American Journal of the Medical Sciences*, April, 1881) reports two cases in which the syphilitic lesion was situated in the upper lobe of a lung, and also shows that the course of syphilitic disease may be both slow and apyretic. Moreover, he states that even after death tubercle cannot always be distinguished from gumma. How is it prior to death? Here the situation is at times relatively easy; often very difficult. Of course, if with the history of an infecting sore we find manifold cutaneous and other lesions, obviously of syphilitic nature, any pulmonary symptoms observed would be apt to be attributed, and properly, to the same diathesis. In such instances, the direct ocular examination of the fauces and pharynx will often reveal signs of great diagnostic value, as, for example, the red symmetrical bands which mark the pillars (Mackenzie), the dry, shiny appearance of the pharynx, and the red and enlarged papillæ on the back of the tongue (Seiler). To this examination may profitably be added an expert one by means of the laryngoscope. Only very lately, Dr. Carl Seiler has published two cases in which this instrument was of much practical value in the diagnosis and treatment (vide *Medical and Surgical Reporter*, April 16, 1881). It would appear from the reading of Dr. Seiler's paper that a like experience has been already communicated to the profession by Dr. John Schnitzler, of Vienna ("Die Lungen Syphilis," 1880). But—supposing the signs of an ulcerative pulmonary affection manifest themselves and progress rapidly, and that neither the history nor examination of the patient reveals the existence of syphilis—how can we determine whether the intra-pulmonary condition is or is not occasioned by syphilis? Frequently, neither rational symptoms nor physical signs will permit us to make the distinction. Almost everything points to the existence of tubercular deposit which has reached the stage of cavities. Even the family history may give increased certainty to this conviction. There may be emaciation, loss of strength, continued fever, profuse purulent expectoration, obstinate cough, and all the accompanying stethoscopic signs of pulmonary phthisis at an advanced period. Treatment alone by mercury and iodide of potash is the touch-stone of this condition when nothing else is, and I have already seen more than one patient, who, in the state described, has been immensely benefited by these remedies, though cod-liver oil and the hypophosphites had been given without appreciable good effects.

In view of these cases what shall we say? Will the mere location of a cavity prove its nature? Evidently not, for I have myself seen the apices affected with destructive lesions, which were only ameliorated by specific medication. And the other pathological conditions already mentioned are not by any means constant at the autopsy. On the contrary, there are patients in whom the advanced phthisical disease is not separable by our most thorough methods of investigation from those on the one hand which are due to syphilis, or on the other, occasioned directly by tubercle. The best we can affirm at the present time is, that there is a pulmonary phthisical affection which appears occasionally in the tertiary stage of syphilis, and which offers by all recognizable signs before death, and at times by those revealed at the autopsy, such close resemblances with advanced tubercular consumption that we are compelled to acknowledge our inability to affirm its precise nature. This position being accepted, it should be recognized that whenever a phthisical predisposition exists already in a patient, and when this patient unfortunately becomes inoculated with syphilis, he has contracted an exceptional morbid imminence to the origin and rapid progress of pulmonary phthisis. Further these dyscrasiæ may march side by side, and post-mortem revelations may indicate merely, as Wagner has shown, that one constitutional disease is at most but modified by the other.

III. *Contagion and Inoculation.*

These two subjects touch one another so closely to-day by the experimental researches of Villemin (1865–66), Marcet, Sanderson, Clark, Fox, and later by Virchow, Schottelius, and Cohnheim, that I naturally wish to consider them together. The medical world in our time has been much divided in regard to the question of direct transmission of pulmonary phthisis. Some there are who can never recognize its existence, always inferring that hereditary predisposition, or bad hygienic conditions are mainly instrumental in its origin and progress. Alongside of this conclusion stand unfortunately numerous undeniable instances in which pulmonary consumption is traceable without question, to breathing an infected atmosphere from a consumptive patient during many weeks or months. Husbands, in previous strong and vigorous health, after caring for phthisical wives, sleeping in the same bed and breathing the same air, have undeniably become consumptives. Wives more frequently, after being placed in similar position as regards affected husbands, have after a time developed tubercular disease, when no explanation other than infection through contact could be satisfactorily offered. These and many analogous examples would suffice to show that in a certain number of well ascertained conditions, the liability to contracting pulmonary phthisis through contact is great. It becomes, therefore, the duty of practitioners to urge the importance of preventive measures. These should be—

(*a*) Separation as far as possible of healthy from affected individuals.

(*b*) Thorough ventilation of all rooms where a husband, wife, sister, or nurse is forcibly obliged to remain many hours of the twenty-four in close attendance upon a phthisical subject.

Is pulmonary phthisis transmissible by food? Chauveau's, Colin's, and other experimental researches prove conclusively :—

1st. That tubercular disease is frequent among cattle, and especially amongst cows and oxen.

2d. That the contact of a diseased animal with a healthy one for a long period, is almost sure to communicate phthisical disease to the other before in a healthy condition. From numerous examples, in which the milk of tubercular cows had been, during many months, the chief source of nutrition of certain children who became pale, emaciated, and finally developed tubercular disease, it is fair to assume that in milk of this origin we have a fruitful source of pulmonary phthisis. Boards of health, properly organized, are, therefore, called upon imperatively to take cognizance of these facts, and to see to it that the milch cows of our cities and towns are unaffected with the "pearl" disease, which is nothing more nor less than a disease similar to pulmonary phthisis in man. Whether the consumption of meat from these diseased animals is capable of producing pulmonary phthisis in man is still debatable, the cooking of such meat appearing to act as an antidote to contagion. The question of the contagion of pulmonary phthisis rests to a great degree upon the possibility of its inoculability. The first experiments of Villemin, Professor at Val de Grace, in 1865, appeared to settle this for a time in the affirmative. Later, Fox, Clark, and Sanderson showed that tubercular disease might be produced by substances not at all specific in nature. Within a year this matter has again been taken up by Cohnheim, and we find ourselves forcibly obliged to admit that, from the inoculation of tubercular or purulent material taken from a phthisical patient, pulmonary phthisis in animals may invariably be developed. It would seem, too, as if the medical world were obliged once more to hold the view first promulgated by Laennec, that pneumonic phthisis and tubercular phthisis are one and the same disease. The investigations of Tappeiner have also indubitably shown that the mere breathing of atomized watery solutions of the sputa of phthisical animals will convey infection to healthy animals. It is, therefore, fairly presumable, in conclusion, from manifold experiments, and more particularly from the latest and most convincing of all, those of Cohnheim, of Leipzig, that pulmonary phthisis in man may be conveyed by—

1st. *The ingesta*, as by the flesh or milk from tubercular cows.

2d. *Inhalation*, as by the particles of sputa carried in the atmosphere directly from phthisical patients.

3d. *Direct inoculation*, as by an abraded surface of a part of the integument through which tubercular matter can be absorbed.

I sum up this brief and imperfect *exposé* of a most important subject by a citation taken from Drake: "A disbelief in the contagion of consumption is highly favourable to the spread of that disease (if it can be really propagated in that mode), inasmuch as in private practice and both in civil and military hospitals no measures of prevention are employed." (Quoted by Clapp: "Is Consumption Contagious?" etc., Boston, 1881.)